occurring in a small percentage of the cases may appear as one of the earliest manifestations of the disease. Of the author's thirty-six cases paresthesia was noted early in the history, appearing simultaneously in feet and hands. Functional nervous disorder was noted in twenty-four cases. In ten cases a spastic and usually an ataxic condition more or less progressive was noted. In three cases complete flaccid paraplegia with loss of knee-jerk and voluntary bowel and bladder control appeared. Girdle sensation was present in the spastic cases contrary to the usual teaching. In three cases there were pathological changes in the cord with a slight sclerosis or degeneration in the lateral and posterior columns of varying intensity and location. There was no marked degeneration of the gray matter of the root zone, of the root ganglia, and of the brain cortex. The degeneration varied at different heights of the cord. It appeared in one case as a focal degeneration, varying decidedly at different heights, not involving a whole system centrifugally or centripetally. It is apt to diffuse itself from one tract to another, but involves the spinal gray as a rule. The whole nerve is not involved. There are two kinds of degenerative processes—an acute, where the nerve sheath becomes swollen, stretched and filled with debris, but not usually associated with an increase of the neuroglia. These lesions of the spinal cord have been attributed to multiple hemorrhages, thickening of the walls of the blood vessels, acute myelitis, primary changes in the gray matter, to anemia, or to a toxic agent. The writer's conclusions are as follows: (1) That there is a well-established relation of diffuse cord degeneration with pernicious anemia. (2) It seems highly probable that the hemolysis and the cord changes are due to the same toxin. (3) While the source of the toxin is unknown the fact that gastro-intestinal disturbance is so common in the disease would lead one to suppose that it is intestinal in origin. (4) The diffuse degeneration of the spinal cord which occurs in conditions without pernicious anemia, does not appear to differ essentially from those of pernicious anemia. (5) It is possible that a common toxin exists as a blood circulating poison in one individual, acts upon the nervous apparatus in another, and coincidentally upon the blood and spinal cord in others. W. B. NOYES.

THE NERVES OF THE HEART. HORACE H. HOPPE, M.D. (Jour. Amer. Med. Asso., May 24, 1902).

A nervous affection of the heart is caused by a faulty condition of the nervous system locally in the heart, the nerves leading to the heart, their center in the medulla, or by a derangement of the nervous system as a whole, as in neurasthenia and hysteria. While the heart is rich in sensory fibers and ganglionic cells, there are no motor fibers nor motor centers. The teaching of the action of the vagus and sympathetic has been modified. The motor stimulus comes from the blood itself. The rhythm of the heart action is automatic, but it can be diminished by influences, chemical, physical or psychical in character, coming either through the sympathetic nerve, or from the vagus. The three kinds of neurosis of the heart are: (1) neurasthenia cardis; (2) tachycardia; and (3) bradycardia. Of neurasthenia cardis we have (a) typical neurasthenia of the heart; (b) arhythmia of the heart; (c) pseudo-angina pectoris; (d) nervous bruit. The first has besides ordinary neurasthenic symptoms, cardiac hypochondria or fear of heart disease based on local precordial sensations, produced by palpitation, irregularities or pain. Fear is a predominent psychic element here as in other forms of neurasthenia. Palpitation is frequent, due to chemical or mechanical conditions of the stomach. It is often associated with a severe sense of constriction of the throat, or dysphonia. The typical neurasthenic heart is weak, irritable and easily exhausted. Dilatation may easily occur. Arhythmia cordis is a condition where irregular or intermittent rhythm occurs even in the absence of all excitement. The most frequent cause is a diseased condition of the coronary arteries or myocarditis. It is not common in neurasthenia with marked vasomotor disturbance, and varies with physical or mental excitement. The nervous ganglia and nerves of the heart being purely sensory, may produce besides disturbances of sensation, distinct pain, either in paroxysms or more or less constant. This is a nervous angina pectoris. Coronary disease is often absent. In most cases the pseudo angina is a neuralgia and entirely functional. It occurs independent of effort, often in the night, associated with other signs of neurasthenia. Tachycardia may occur as a pure neurosis, independent of all signs of dilatation or other disease of the heart. It may be due to irritation of the sympathetic. Bradycardia is rare as a pure neurosis; it may be reflex from gastro-intestinal disturbances, and occurs in paroxysms. It is best treated by sedatives, such as bromides, asafetida and valerian.

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ACUTE ASCENDING PARALYSIS IN BASEDOW'S DISEASE. M. ROSENFELD (Berlin. klin. Woch., June 9, 1902).

Regarding paralysis of the extremities occurring in Basedow's disease, to which Charcot and Mackinsie called attention, as occurring without loss of sensation, weakness of the bladder or muscular atrophy, this author relates the history of a case where the cachectic period of the disease began with an acute ascending paralysis, following a course similar to that of Landry's disease. Although the patient had been ill for two years, suffering during one year with exophthalmos and extreme nervousness, he had continued at work without interruption. On the day of the attack he experienced nausea and headache in the morning and later in the day lumbar pains and a feeling of weight in the lower extremities. The next day pain and weakness compelled the patient to stop work, but there was no fever and the cranial nerves and sensation were normal, as well as the movements of the upper extremities, although walking or standing was impossible. The right leg was absolutely powerless and the patellar reflex absent, but the left leg could be moved a little and a slight reflex obtained on that side. The bladder and rectum remained normal and the chief complaint was of lumbar pain radiating to the extremities. After a few hours the power of sitting erect was lost, while the arms could only be moved with difficulty, and before night there was complete paralysis of the arms and the head could not be moved. There was slight motion in the fingers but the bladder was paralyzed, although respiration continued normal without loss of consciousness. The next day voluntary motion of the extremities returned but a rapid loss of weight ensued, together with an increase of all the usual symptoms of Basedow's disease and a permanent loss of muscular power. This attack was followed by two others which were neither of them as severe as the first. Hysteria may be excluded on the following grounds; the prodromata, the severe collapse at the outset of the paralytic attack, the paralysis gradually ascending, the disappearance of the right patellar reflex during the paroxysm and the existence of a light paresis of the right leg with diminished reflex after the attack.

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